UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Ex parte CHRISTOPHER SILVIA and WEIFENG YU

Appeal No. 2005-2599
Application No. 09/623,304

ON BRIEF

Before SCHEINER, ADAMS and GRIMES, Administrative Patent Judges.

SCHEINER, Administrative Patent Judge.

DECISION ON APPEAL

This appeal involves claims to a nucleic acid encoding a polypeptide monomer of an alpha subunit of a human inward rectifier potassium channel. The examiner has rejected the claims under 35 U.S.C. §§ 101 and 112, first paragraph, as lacking both utility and enablement, and under 35 U.S.C. § 112, second paragraph, as indefinite. We have jurisdiction under 35 U.S.C. § 134. We affirm the rejections for lack of utility and enablement, and do not reach the rejection for indefiniteness.

BACKGROUND

Potassium channels "allow the flow of potassium in and/or out of [a] cell under certain conditions" and "are regulated, e.g., by calcium sensitivity, voltage-gating, second messengers, extracellular ligands, and ATP-sensitivity." Specification, page 1.

"Potassium channels are involved in a number of physiological processes, including regulation of heartbeat, dilation of arteries, release of insulin, excitability of nerve cells, and regulation of renal electrolyte transport . . . [and] are thus found in a wide variety of animal cells such as nervous, muscular, glandular, immune, reproductive, and epithelial tissue." Id.

There are eight families of potassium channels, based on predicted structural and functional similarities. Id., page 2. "The Kir family of inward rectifier potassium channels includes both heteromeric and homomeric channels that are typically composed of four [alpha] subunits . . . Inward rectifier channels primarily allow potassium influx, with little potassium outflux" (id.), and "have significant roles in maintaining the resting potential and in controlling excitability of a cell" (id., page 6).

The present invention is directed to nucleic acids encoding a human inward rectifier potassium channel alpha subunit designated hKir5.1. The hKir5.1 subunit "was expressed according to standard methodology, . . . [and] its ability to form heteromeric potassium channels with inward rectifier activity" was detected by measuring changes in current magnitude across the membranes of cells expressing hKir5.1 alone or in combination with another alpha subunit, hKir4.1 (id., page 57). In addition, "RNA expression of hKir5.1 was examined in human tissues" and "[t]he distribution of hKir5.1 indicates channels comprising this subunit may play an important role in the function of the kidney, thyroid, pancreas[ ], and salivary gland" (id., page 58).

According to the specification, "chromosomal localization of hKir5.1 can be used to identify diseases caused by and associated with hKir5.1" and "detecting hKir5.1 [is] also useful for examining the role of hKir5.1 in channel diversity and modulation of

channel activity" (id., page 9). Finally, "biologically active hKir5.1 . . . provides a means for assaying for inhibitors and activators of heteromeric inward rectifier potassium channels that comprise hKir5.1 subunits" (id.). According to the specification, "[s]uch activators and inhibitors are useful as pharmaceutical agents for treating disorders such as hypertension, acute renal failure, chronic renal failure, diabetes insipidus, diabetic nephropathy, hypothyroidism, hyperthyroidism, goiter, hypoparathyroidism, hyperparathyroidism, pancreatic insufficiency, diabetes, cystic fibrosis, sialorrhea, and salivary insufficiency" (id.).

THE CLAIMS

Claims 1-4, 6 and 7 are pending in the application. Claim 1 is representative of the subject matter on appeal:

> 1. An isolated nucleic acid encoding a polypeptide monomer comprising an alpha subunit of a potassium channel, the polypeptide monomer:
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> (i) forming, with at least one additional Kir alpha subunit, a potassium channel having the characteristic of inward rectification; and
>
> (ii) encoded by a nucleic acid that selectively hybridizes under highly stringent hybridization conditions to a nucleotide sequence of SEQ ID NO:2, wherein the stringent conditions comprise incubation at 42°C in a solution comprising 50% formamide, 5 x SSC and 1% SDS or an incubation at 65°C in a solution comprising 5 x SSC and 1% SDS at 65°C with a wash in 0.2 x SSC and 0.1% SDS.

DISCUSSION

Utility and Enablement

The examiner rejected all of the pending claims as lacking a disclosed utility sufficient to satisfy 35 U.S.C. § 101. The examiner also rejected all of the claims under 35 U.S.C. § 112, first paragraph, for lack of enablement, but that rejection is merely a

corollary of the finding of lack of utility (Answer, page 20). Therefore, our conclusion with respect to the § 101 issue also applies to the § 112 issue.

The examiner bears the initial burden of showing that a claimed invention lacks patentable utility. See In re Brana, 51 F.3d 1560, 1566, 34 USPQ2d 1436, 1441 (Fed. Cir. 1995) ("Only after the PTO provides evidence showing that one of ordinary skill in the art would reasonably doubt the asserted utility does the burden shift to the applicant to provide rebuttal evidence sufficient to convince such a person of the invention's asserted utility.").

The U.S. Court of Appeals for the Federal Circuit recently addressed the utility requirement in the context of a claim to DNA. See In re Fisher, 421 F.3d 1365, 76 USPQ2d 1225 (Fed. Cir. 2005). The Fisher court interpreted Brenner v. Manson, 383 U.S. 519, 148 USPQ 689 (1966), as rejecting a "de minimis view of utility." 421 F.3d at 1370, 76 USPQ2d at 1229. The Fisher court held that § 101 requires a utility that is both substantial and specific. Id. at 1371, 76 USPQ2d at 1229. The court held that disclosing a substantial utility means "show[ing] that an invention is useful to the public as disclosed in its current form, not that it may be useful at some future date after further research. Simply put, to satisfy the 'substantial' utility requirement, an asserted use must show that that claimed invention has a significant and presently available benefit to the public." Id., 76 USPQ2d at 1230.

The court held that a specific utility is "a use which is not so vague as to be meaningless." Id. In other words, "in addition to providing a 'substantial' utility, an asserted use must show that that claimed invention can be used to provide a well-defined and particular benefit to the public." Id.

The Fisher court held that none of the uses asserted by the applicant in that case were either substantial or specific. The uses were not substantial because "all of Fisher's asserted uses represent merely hypothetical possibilities, objectives which the claimed ESTs, or any EST for that matter, could possibly achieve, but none for which they have been used in the real world." Id. at 1373, 76 USPQ2d at 1231. "Consequently, because Fisher failed to prove that its claimed ESTs can be successfully used in the seven ways disclosed in the '643 application, we have no choice but to conclude that the claimed ESTs do not have a 'substantial' utility under § 101." Id. at 1374, 76 USPQ2d at 1232.

"Furthermore, Fisher's seven asserted uses are plainly not 'specific.' Any EST transcribed from any gene in the maize genome has the potential to perform any one of the alleged uses. . . . Nothing about Fisher's seven alleged uses set the five claimed ESTs apart from the more than 32,000 ESTs disclosed in the '643 application or indeed from any EST derived from any organism. Accordingly, we conclude that Fisher has only disclosed general uses for its claimed ESTs, not specific ones that satisfy § 101." Id.

Utility is determined as of the effective filing date of the application. See In re Brana, 51 F.3d at 1567 n.19, 34 USPQ2d at 1441 n.19. Here, the specification discloses that the protein encoded by the claimed nucleic acid is a subunit of an inward rectifier potassium channel preferentially expressed in kidney, thyroid, pancreas and salivary gland tissues, and this disclosure was confirmed by expression of the protein. The relevant question with respect to utility, then, is whether a specific and substantial utility for hKir5.1 (or any other inward rectifier potassium channel, for that matter) was disclosed in the specification or well known in the art as of the effective filing date of the present application.

Appellants argue essentially that an asserted utility is specific, substantial and credible "when applicants disclose a 'specific biological activity' and reasonably correlate that activity to a 'disease condition'" (Brief, page 9). Appellants argue that the present specification "identifies the nucleic acid and amino acid sequences of human Kir5.1, demonstrates the expression pattern of Kir5.1, . . . and illustrates the electrophysiological characteristics of the Kir5.1 channels" and "further discloses a 'disease condition' (i.e., disorders . . . including hypertension, renal failure, and the like) that correlates with a 'biological activity' (i.e., the opening and closing of a Kir5.1 channel)" (id., page 10). Along the same lines, appellants argue that "the Kir family of inward rectifier potassium channels is known to be involved in regulating potassium flow across [the] cell membrane and therefore regulating cell resting potential and excitability" (id., page 8), thus, "one of skill in the art would expect a Kir5.1 potassium channel to play an important role in the proper physiological function of the tissues where it is specifically expressed" (id.). Consequently, according to appellants, "compounds capable of modulating the activity of a Kir5.1 potassium channel can be used for treating conditions and disorders in these tissues that are caused by abnormal potassium influx" (id.).

Nevertheless, the examiner found the specification's disclosure of utility to be inadequate because it "fails to provide any factual evidence that this specific Kir5.1 subunit is associated with any particular disease, condition, or physiological process other than a general regulation of cell excitability" under unspecified conditions (Answer, page 9). According to the examiner, "mere expression of the protein in a tissue is not . . . a showing of a role of the Kir5.1 DNA or protein in hypertension, acute renal failure,

chronic renal failure, diabetes insipidus, diabetic nephropathy, hypothyroidism, hyperthyroidism, goiter, hypoparathyroidism, hyperparathyroidism, pancreatic insufficiency, diabetes, cystic fibrosis, sialorrhea, [or] salivary insufficiency" (id., page 12).

In addition, the examiner cited a number of literature references as evidence that "the human Kir5.1 polynucleotide and polypeptide . . . [were] not well characterized" as of the effective filing date of the application (Answer, page 6). For example, Tanemoto[1] teaches that "[t]he inwardly rectifying potassium (Kir) channel family is now known to possess more than 20 members, which can be classified into four major subfamilies . . . [which] play pivotal roles in determining the resting membrane potential (Kir2.0), in G protein- or intracellular metabolism-dependent regulation of cell excitability (Kir3.0 and Kir6.0, respectively), and in transporting K^+ ions in epithelial tissues and glial cells (Kir1.1 and Kir4.0). [However,] Kir5.1 does not belong to any of these subfamilies and its physiological roles are unknown" (Tanemoto, page 587). Similarly, Pessia[2] teaches that "[t]he functional role of [ ] heteromeric Kir4.0-Kir5.1 channels remains unclear" (Pessia, page 359), even though "recent studies have shown that Kir4.1-Kir5.1 heteromeric channels exist in vivo in renal tubular epithelia . . . [and] [t]here is [ ] an emerging role for these channels in the pH-dependent regulation of K^+ fluxes and acid-base homeostasis" (Pessia, page 359).

The examiner concluded that "the instant specification does not disclose a 'real world' use for [h]Kir5.1" (id., page 7), and "[s]ignificant further experimentation would be

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[1] Tanemoto et al., Journal of Physiology, Vol. 525, No. 3, pp. 587-592 (2000).
[2] Pessia et al., Journal of Physiology, Vol. 532, No. 2, pp. 359-367 (2001).

required . . . so that [hKir5.1] could be readily used in a real world sense" (id., page 11). "[T]herefore, [hKir5.1] lacks utility in currently available form" (id., page 10).

We agree with the examiner that the claimed nucleic acids do not have utility simply because they encode an alpha subunit of an inward rectifier potassium channel. Even the observation that hKir5.1 is preferentially expressed in kidney, thyroid, pancreas and salivary gland tissues does not reveal a significant, presently available, and well-defined benefit to the public because no "conditions owing to diminished or aberrant expression" of hKir5.1 are disclosed in the specification (Specification, page 54). Nor have appellants pointed to any evidence outside of the specification, and publicly available before this application's effective filing date, that would have allowed a person skilled in the art to use the nucleic acid encoding hKir5.1 in a specific and substantial way. We therefore conclude that the mere identification of the protein encoded by the claimed nucleic acids as a subunit of an inward rectifier potassium channel does not satisfy the utility requirement of 35 U.S.C. § 101.

The rejections of claims 1-4, 6 and 7 under 35 U.S.C. §§ 101 and 112, first paragraph, are affirmed.

Summary

The specification does not disclose a specific and substantial utility for the claimed nucleic acids, as required by 35 U.S.C. § 101. We therefore affirm the examiner's rejection of claims 1-4, 6 and 7 under § 101, and the corresponding rejection under § 112, first paragraph. Our decision constitutes a disposition of all the claims on appeal, accordingly, we do not reach the indefiniteness rejection of the claims under 35 U.S.C. § 112, second paragraph.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 CFR § 1.136(a).

AFFIRMED

Toni R. Scheiner)
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